

# VENTRICULAR REPOLARIZATION EVALUATION FROM SURFACE ECG FOR IDENTIFICATION OF THE PATIENTS WITH INCREASED MYOCARDIAL ELECTRICAL INSTABILITY

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**Abstract –** In order to reveal the possible correlation between the level of myocardial electrical instability assessed at Holter monitoring and certain ECG parameters characterizing ventricular repolarization 24-hours ECG recordings were analyzed in 91 patients with different grades of ventricular arrhythmias.

The following parameters were calculated: RT-interval (RT) duration and variability, RT apex interval (RTa) duration and variability, areas of the first and second half of T-wave (S1, S2) and maximal rise and fall slopes of T-wave (k1, k2). An original signal processing algorithm for ECG was developed for that purpose.

The results of the study suggest that complex analysis of certain T-wave parameters, as well as RT interval variability can be a useful tool for identification of patients at increased risk of sudden death.

**Keywords –** Sudden cardiac death, T-wave morphology, RT interval variability

## I. INTRODUCTION

Sudden cardiac death (SD) is one of the major problems in modern cardiology. SD accounts for about an half of all deaths in most common cardiac diseases, and its occurrence in the whole population is 2 per thousand a year. Numerous invasive and non-invasive tests for risk stratification are used in clinical practice, as an identification of high-risk patients in time is a desirable therapeutic goal. Significant reduction of as high as 30-40 % annual mortality rate by appropriate treatment has been reported.

During the last decade a lot of attention has been paid to the investigation of the ventricular repolarization phase. The parameters, reflecting left ventricular repolarization prolongation and spatial inhomogeneity, mainly different modifications of QT interval dispersion (QTD) measured from the standard 12-lead ECG, have been reported to possess considerable prognostic value for predicting sudden death not only in post-myocardial infarction patients [1-3], but also in patients with other forms of coronary artery disease (CAD) [4,5] as well as in other pathological heart conditions [6, 7]. Two [8, 9] large-scale epidemiological studies published recently have shown that the QT interval prolongation and dispersion are independent predictors of outcome in large populations.

During recent years a dynamic property of ventricular repolarization - QT interval variability (QTV) - has

undergone even more intensive investigation in order to assess its utility for high risk patients' identification.

Clinical and prognostic significance of temporal variations of repolarization during 24 hours has been evaluated since the middle of nineties still obtaining controversial results [10-12]. It has been demonstrated that in healthy subjects heart rate exhibits substantial beat-to-beat variability (HRV), which is mirrored in the instantaneous QT interval. In several pathological conditions where the HRV is depressed due to withdrawal of parasympathetic tone, the QT interval fluctuates widely without any discernible relation to instantaneous heart rate. The increased variability of the QT interval in response to heart rate changes might reflect unstable ventricular repolarization, which acts as a substance for ventricular arrhythmias [13]. Algra et al. [14] demonstrated that elevated ( $>25$  ms) QTV over 24 hours was associated with more than twofold risk of sudden death compared to intermediate variation (20-25 ms) values. Atiga et al. [15] reported that temporal QT interval dispersion was the only clinical variable (in addition all patients underwent spatial QTD measurement, HRV, late potentials and T- wave alternans registration, programmed ventricular stimulation) that identified SD patients. The prognostic significance of this parameter is augmented by the circumstance that the increase in QT temporal variability already appears at early stages of the disease. So, Berger et al [16] demonstrated in ischemic and non-ischemic dilated cardiomyopathy patients that significantly greater QTV prevailed among minimally symptomatic patients with little or no further increase among the patients with more severe stages of heart failure.

The surface T-wave is the result of spatial heterogeneity in the action potential duration of cardiac cells. During recent years several new approaches for T-wave morphology analysis have been proposed as an alternative technique for high-risk patients identification, including the area under T(U) wave determination [17], the technique of principal components analysis [18, 19], determination of spatial and temporal variations of T-wave morphology and repolarization waveform direction [20], etc. According to the existing data [21], these variables seem to be independent of heart rate and QT interval duration. At the same time, it is generally accepted that the computer-aided T-wave shape analysis is still in the experimental phase, so

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further effort is required in order to assess the clinically relevant contents of the T-wave morphology [22].

The aim of the study was to compare predictive value of different parameters calculated on the basis of T-wave in patients with ventricular arrhythmia. A novel T-wave detection algorithm for long term ECG signal evaluation is proposed.

## II. MATERIALS AND METHODS

Ninety-one persons, included in the study, were divided into four groups according to the maximal Lown grade ventricular arrhythmia at Holter monitoring.

The groups were as following:

Group O – grade 0 ectopy – no ventricular premature beats at 24-hours ECG recording;

Group 3 A – grade 3 A ectopy – polymorphic ventricular premature beats;

Group 4A - grade 4A ectopy – ventricular couplets;  
Group 4B- grade 4B ectopy – ventricular tachycardia of 3 or more beats.

Recordings were divided into four above-mentioned groups by experienced cardiologist decision. Group 3A included 33 patients (13 female and 20 male, mean age  $52 \pm 17$ ), Group 4A - 18 patients (10 female and 8 male, mean age  $59 \pm 12$ ) and group 4B 13 patients (2 female and 11 male, mean age  $56 \pm 18$ ). The fourth Group O (27 patients, 20 female and 7 male, mean age  $29 \pm 19$ ) was the reference group.

ECG was recorded using a 3-channel, 24-hour ECG recorder (model RZ-152, Rozinn), frequency of discretization 180Hz, resolution 10 bits. The full disclosure of 24-hour signal from the same ECG channel (channel 0) for every patient was chosen for further analysis.

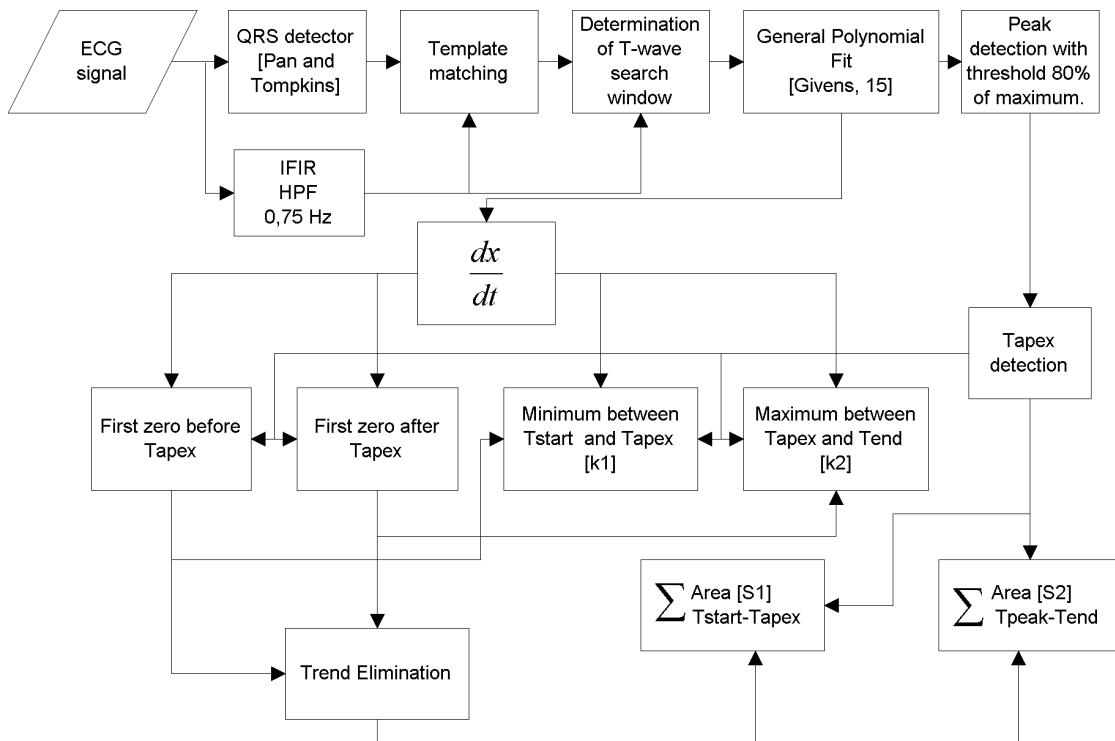


Figure 1. Block diagram of ECG signal pre-processing algorithm

Analysis of the 24-hour recordings was performed with a special algorithm presented on the Figure 1. The locations of QRS complexes were first detected by algorithm proposed by Tompkins [23]. Thereafter a template matching subroutine was used to extract the normal complexes from abnormal ones. T-wave parameters were calculated with the help of polynomial fit (Givens algorithm, order 15) of T-wave. Before the curve fitting the signal was filtered with the high-pass filter to remove baseline wandering. The cut-off frequency for the filter was 0.75Hz. The interpolated FIR (IFIR) filter algorithm was used for that purpose. After

that the approximate T-wave search window was determined from consequent RR interval. After completion of the curve fitting procedure all peaks above 80% level inside the window were detected and the first valid peak was chosen to be T-wave apex.

Simultaneously the first derivative of polynomial fit of T-wave was calculated and the first zero before and after the T-apex were detected and named consequently as T-start and T-end. Thereafter the maximum of derivative between T-start and T-apex was calculated as the maximum slope of T-wave rising phase (k1). A similar procedure was

performed for maximal slope of falling phase of T-wave (k2) calculation. After the removal of possible remaining of DC shift from T-wave the areas under the wave were calculated separately for rising and falling phase and named consequently as S1 and S2. The algorithm was designed in the signal processing environment LabVIEW.

For the final analysis of differences between the groups two parameters were calculated for every patient from 24-hour signal. The mean value of every parameter was calculated and also the standard deviation of a population

based on a sample (STD) that represents the variability of the parameter.

### III. RESULTS

The results of the work are presented in Table 1 and 2. The Student's t-test was performed for statistical comparison of differences between the groups and calculated significance levels (p values) are presented in Table 2.

TABLE I  
Calculated mean and standard deviation values of parameters for different groups

	0 N=27		3A N=33		4A N=18		4B N=13	
	Mean	STD	Mean	STD	Mean	STD	Mean	STD
<b>RT mean (ms)</b>	204,95	18,38	208,08	27,4	218,53	25,85	216,42	33,55
<b>RT STD</b>	24,8	8,33	27,41	9,8	31,91	14,11	33,55	12,63
<b>RTa mean (ms)</b>	292,94	15,55	299,06	22,97	305,67	21,14	301,11	28,68
<b>RTa STD</b>	28,34	7,13	30,84	7,74	32,87	9,83	33,85	9,69
<b>s1 mean (a.u.)</b>	47091,6	19413,11	34879,24	34628,6	25555,39	13491,34	23128,59	12300,6
<b>s1 STD</b>	18795,04	5892,21	14510,83	8581,11	12452,83	4556,57	12807,13	6841,65
<b>s2 mean (a.u.)</b>	34689,58	11945,48	25927,28	16638,12	21021,34	11021,17	17653,33	8319,72
<b>s2 STD</b>	13238,94	3217,31	10539,03	3667,08	9988,96	4127,34	9133,57	3277,52
<b>k1 mean (a.u.)</b>	373,11	131,78	298,66	194,22	222,66	92,26	235,36	110,29
<b>k1 STD</b>	276,57	123,59	214,84	79,46	175,05	67	194,03	86,53
<b>k2 mean (a.u.)</b>	614,96	258,75	384,18	178,76	328,13	163,17	302,31	129,14
<b>k2 STD</b>	286,89	126,1	213,82	78,14	198,64	102,89	186,32	96,68

TABLE II  
Statistical comparison of calculated parameters with two-tailed t-test

	0&3A	0&4A	0&4B	3A&4A	3A&4B	4A&4B	0,3A&4A,4B	0,3A,4A&4B
	p1	p2	p3	p4	p5	p6	p7	p8
<b>RT mean</b>	0,60100	0,06350	0,26600	0,18500	0,43400	0,85200	0,03660	0,24000
<b>RT STD</b>	0,26800	0,06490	0,03570	0,23900	0,13200	0,73800	0,01070	0,06250
<b>RTa mean</b>	0,22500	0,03630	0,35100	0,30700	0,82100	0,63100	0,07350	0,37700
<b>RTa STD</b>	0,19800	0,10300	0,08280	0,45500	0,33000	0,78500	0,03890	0,12500
<b>s1 mean</b>	0,09090	0,00007	0,00003	0,17800	0,09670	0,60700	0,00028	0,00226
<b>s1 STD</b>	0,02600	0,00020	0,01280	0,26900	0,48600	0,87200	0,00392	0,10300
<b>s2 mean</b>	0,02110	0,00031	0,00001	0,21300	0,03060	0,34000	0,00011	0,00064
<b>s2 STD</b>	0,00354	0,00821	0,00098	0,64000	0,21700	0,52500	0,00633	0,02040
<b>k1 mean</b>	0,08340	0,00005	0,00161	0,06450	0,17300	0,73800	0,00021	0,02860
<b>k1 STD</b>	0,03000	0,00093	0,01980	0,06470	0,46000	0,51500	0,00128	0,11500
<b>k2 mean</b>	0,00028	0,00004	0,00001	0,26400	0,09380	0,62700	0,00004	0,00119
<b>k2 STD</b>	0,01190	0,01360	0,00888	0,58900	0,37100	0,73600	0,01060	0,05590

### IV. DISCUSSION AND CONCLUSIONS

The results of our study demonstrate that certain T-wave morphology parameters, such as areas S1, S2 and slopes k1, k2 possess significant prognostic capabilities for predicting potentially life-threatening arrhythmias at Holter monitoring. The area of the falling phase of T-wave (S2) seems to be the most sensitive parameter in this set. The

only insignificant difference for S1 was recorded between groups 4A and 4B, but nevertheless the strong tendency of S1 decrease at higher arrhythmias grades was revealed. The temporal parameters RT and RTa differ significantly in Group 0 and 4 patients.

The utilization of the study results - non-invasive analysis of temporal ventricular repolarization heterogeneity and certain aspects of T-wave morphology - provides an

important tool for sudden death risk stratification and assessment of antiarrhythmic drug treatment benefits/drawbacks in patients with CAD.

These methods together with other non-invasive methods of ventricular repolarization inhomogeneity assessment may turn to be useful for identification of patients with ventricular arrhythmias for invasive electrophysiologic study, catheter ablation and implantable devices treatment.

Thus, the results of our study demonstrate that several parameters characterizing ventricular repolarization phase on surface ECG appear to have strong correlation with the level of myocardial electrical instability assessed at Holter monitoring. The determination of the clinical significance of these parameters requires further investigations.

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